

## EAST Search History

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	10	Tani near2 Kousuke	US-PGPUB	OR	ON	2006/04/05 10:59
L3	1	EP3 same autoimmune	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 13:04
L4	7	EP3 same allerg\$	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 13:06
L5	29	EP3.ti,ab.	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 13:15
L6	6	EP3.ti,ab. and (allerg\$ or asthma)	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 13:07
L7	1619	"prostaglandin E2"	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 13:16
L8	849	"prostaglandin E2" and (allerg\$ or asthma)	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 13:16
L9	49	"prostaglandin E2" same (allerg\$ or asthma)	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 13:16
L10	10	"prostaglandin E2" with (allerg\$ or asthma)	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 13:16
S1	2	"6642266".pn. "4386031".pn.	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 10:59
S2	2	EP3 with allerg\$	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 13:04
S3	7	EP3 same allerg\$	US-PGPUB; USPAT; EPO	OR	ON	2006/04/05 13:04
S4	211	strong.in.	US-PGPUB	OR	ON	2006/03/24 07:19
S5	1	strong.in. and cox-2.ti.	US-PGPUB	OR	ON	2006/03/24 07:29
S6	0	("2868691" "3095355" "3965143" "3985791" "4301146" "4692464" "4863961" "5624959" "5723493" "5753700" "6013673").PN.	US-PGPUB	OR	ON	2006/03/24 07:29
S7	11	("2868691" "3095355" "3965143" "3985791" "4301146" "4692464" "4863961" "5624959" "5723493" "5753700" "6013673").PN.	US-PGPUB; USPAT; EPO	OR	ON	2006/03/24 07:31

## EAST Search History

S8	1	"5157052".pn.	US-PGPUB; USPAT; EPO	OR	ON	2006/03/24 07:31
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USPAT2

NEWS	5	JAN	13	IPC 8 searching in IFIPAT, IFIUDB, and IFICDB
NEWS	6	JAN	13	New IPC 8 SEARCH, DISPLAY, and SELECT enhancements added to INPADOC
NEWS	7	JAN	17	Pre-1988 INPI data added to MARPAT
NEWS	8	JAN	17	IPC 8 in the WPI family of databases including WPIFV
NEWS	9	JAN	30	Saved answer limit increased
NEWS	10	JAN	31	Monthly current-awareness alert (SDI) frequency added to TULSA
NEWS	11	FEB	21	STN AnaVist, Version 1.1, lets you share your STN AnaVist visualization results
NEWS	12	FEB	22	Status of current WO (PCT) information on STN
NEWS	13	FEB	22	The IPC thesaurus added to additional patent databases on STN
NEWS	14	FEB	22	Updates in EPFULL; IPC 8 enhancements added
NEWS	15	FEB	27	New STN AnaVist pricing effective March 1, 2006
NEWS	16	FEB	28	MEDLINE/LMEDLINE reload improves functionality
NEWS	17	FEB	28	TOXCENTER reloaded with enhancements
NEWS	18	FEB	28	REGISTRY/ZREGISTRY enhanced with more experimental spectral property data
NEWS	19	MAR	01	INSPEC reloaded and enhanced
NEWS	20	MAR	03	Updates in PATDPA; addition of IPC 8 data without attributes
NEWS	21	MAR	08	X.25 communication option no longer available after June 2006
NEWS	22	MAR	22	EMBASE is now updated on a daily basis
NEWS	23	APR	03	New IPC 8 fields and IPC thesaurus added to PATDPAFULL
NEWS	24	APR	03	Bibliographic data updates resume; new IPC 8 fields and IPC thesaurus added in PCTFULL
NEWS	25	APR	04	STN AnaVist \$500 visualization usage credit offered

NEWS EXPRESS    FEBRUARY 15 CURRENT VERSION FOR WINDOWS IS V8.01a, CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP), AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005. V8.0 AND V8.01 USERS CAN OBTAIN THE UPGRADE TO V8.01a AT <http://download.cas.org/express/v8.0-Discover/>

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\* \* \* \* \* STN Columbus \* \* \* \* \*

FILE 'HOME' ENTERED AT 11:56:16 ON 05 APR 2006

=> file medline

COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	0.42	0.42

FILE 'MEDLINE' ENTERED AT 11:57:37 ON 05 APR 2006

FILE LAST UPDATED: 4 APR 2006 (20060404/UP). FILE COVERS 1950 TO DATE.

On December 11, 2005, the 2006 MeSH terms were loaded.

The MEDLINE reload for 2006 is now (26 Feb.) available. For details on the 2006 reload, enter HELP RLOAD at an arrow prompt (=>). See also:

<http://www.nlm.nih.gov/mesh/>  
[http://www.nlm.nih.gov/pubs/techbull/nd04/nd04\\_mesh.html](http://www.nlm.nih.gov/pubs/techbull/nd04/nd04_mesh.html)  
[http://www.nlm.nih.gov/pubs/techbull/nd05/nd05\\_med\\_data\\_changes.html](http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_med_data_changes.html)  
[http://www.nlm.nih.gov/pubs/techbull/nd05/nd05\\_2006\\_MeSH.html](http://www.nlm.nih.gov/pubs/techbull/nd05/nd05_2006_MeSH.html)

OLDMEDLINE is covered back to 1950.

MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the MeSH 2006 vocabulary.

This file contains CAS Registry Numbers for easy and accurate substance identification.

```
=> s EP3 (L) (allergy or asthma)
      654 EP3
      42129 ALLERGY
      90571 ASTHMA
L1      7 EP3 (L) (ALLERGY OR ASTHMA)
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=> d 1-7 bib abs
```

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L1  ANSWER 1 OF 7      MEDLINE on STN
AN  2005516639      IN-PROCESS
DN  PubMed ID: 16189372
TI  Evaluation of selective prostaglandin E2 (PGE2) receptor agonists as
    therapeutic agents for the treatment of asthma.
AU  Chung K F
CS  National Heart and Lung Institute, Imperial College London, London SW3
    6LY, UK.. f.chung@imperial.ac.uk
SO  Science's STKE [electronic resource] : signal transduction knowledge
    environment, (2005 Sep 27) Vol. 2005, No. 303, pp. pe47. Electronic
    Publication: 2005-09-27.
    Journal code: 100964423. E-ISSN: 1525-8882.
CY  United States
DT  Journal; Article; (JOURNAL ARTICLE)
LA  English
FS  NONMEDLINE; IN-PROCESS; NONINDEXED; Priority Journals
ED  Entered STN: 20050929
    Last Updated on STN: 20051215
AB  Prostaglandin E2 (PGE2) released in asthmatic airways has bronchodilator
    properties and inhibits allergen-induced bronchoconstriction and release
    of inflammatory mediators. Although considered as a potential treatment
    for asthma, PGE2 also has some proinflammatory properties. PGE2
    acts through four different receptor subtypes (EP1, EP2, EP3,
    and EP4) that may explain some of PGE2's diverse effects. In a mouse
    model of allergic inflammation in which the four receptors were
    individually deleted, only EP3(-/-) mice showed an enhancement
    of inflammation, whereas an EP3 agonist was inhibitory, with
    PGE2 being inactive. Thus, EP3 agonists may lead to a new
    approach for the treatment of asthma. However, other PGE2
    receptor subtypes may also have beneficial effects, and a greater
    understanding of the signaling pathways of these receptor subtypes will
    help to clarify the role of these receptors in asthma.

L1  ANSWER 2 OF 7      MEDLINE on STN
AN  2005209441      MEDLINE
DN  PubMed ID: 15806106
TI  Suppression of allergic inflammation by the prostaglandin E receptor
    subtype EP3.
AU  Kunikata Tomonori; Yamane Hana; Segi Eri; Matsuoka Toshiyuki; Sugimoto
    Yukihiko; Tanaka Satoshi; Tanaka Hiroyuki; Nagai Hiroichi; Ichikawa
    Atsushi; Narumiya Shuh
CS  Department of Pharmacology and Faculty of Medicine and Graduate School of
    Pharmaceutical Sciences, Kyoto University, Kyoto 606-8501, Japan.
SO  Nature immunology, (2005 May) Vol. 6, No. 5, pp. 524-31. Electronic
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Publication: 2005-04-03.  
Journal code: 100941354. ISSN: 1529-2908.

CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 200505  
ED Entered STN: 20050422  
Last Updated on STN: 20050517  
Entered Medline: 20050516

AB Prostaglandins, including PGD(2) and PGE(2), are produced during allergic reactions. Although PGD(2) is an important mediator of allergic responses, aspirin-like drugs that inhibit prostaglandin synthesis are generally ineffective in allergic disorders, suggesting that another prostaglandin-mediated pathway prevents the development of allergic reactions. Here we show that such a pathway may be mediated by PGE(2) acting at the prostaglandin E receptor EP3. Mice lacking EP3 developed allergic inflammation that was much more pronounced than that in wild-type mice or mice deficient in other prostaglandin E receptor subtypes. Conversely, an EP3-selective agonist suppressed the inflammation. This suppression was effective when the agonist was administered 3 h after antigen challenge and was associated with inhibition of allergy-related gene expression. Thus, the PGE(2)-EP3 pathway is an important negative modulator of allergic reactions.

L1 ANSWER 3 OF 7 MEDLINE on STN  
AN 2004537201 MEDLINE  
DN PubMed ID: 15257985

TI Augmentation of bovine airway smooth muscle responsiveness to carbachol, KCl, and histamine by the isoprostane 8-iso-PGE2.

AU Catalli Adriana; Janssen Luke J

CS Firestone Institute for Respiratory Health and Father Sean O'Sullivan Research Center, St. Joseph's Hospital, Hamilton, Ontario, Canada.

SO American journal of physiology. Lung cellular and molecular physiology, (2004 Nov) Vol. 287, No. 5, pp. L1035-41. Electronic Publication: 2004-07-16.

Journal code: 100901229. ISSN: 1040-0605.

CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 200411  
ED Entered STN: 20041029  
Last Updated on STN: 20041219  
Entered Medline: 20041119

AB Isoprostanes are generated during periods of oxidative stress, which characterize diseases such as asthma and cystic fibrosis. They also elicit functional responses and may therefore contribute to the pathology of these diseases. We set out to examine the effects of isoprostanes on airway responsiveness to cholinergic stimulation. Muscle bath techniques were employed using isolated bovine tracheal smooth muscle. 8-Isoprostaglandin E2 (8-iso-PGE2) increased tone directly on its own, although the magnitude of this response, even at the highest concentration tested, was only a fraction of that evoked by KCl or carbachol. More importantly, though, pretreatment of the tissues with 8-iso-PGE2 (10 microM) markedly augmented responses to submaximal and even subthreshold concentrations of KCl, carbachol, or histamine, whereas maximal responses to these agents were unaffected by the isoprostane. The augmentative effect on cholinergic responsiveness was mimicked by PGE2 (0.1 microM) and by the FP agonists PGF2 (0.1 microM) and fluprostenol (0.1 microM), but not by the EP3 agonist sulprostone (0.1 microM) or the TP agonist U-46619 (0.1 microM). Antagonists of EP1 receptors (AH-6809 and SC-19920, 10 microM) and TP receptors (ICI-192605, 1 microM) had no effect on 8-iso-PGE2-induced augmentation of cholinergic

responsiveness. We conclude that 8-iso-PGE2 induces nonspecific airway smooth muscle hyperresponsiveness through a non-TP non-EP prostanoid receptor.

L1 ANSWER 4 OF 7 MEDLINE on STN  
AN 2004231827 MEDLINE  
DN PubMed ID: 15131569  
TI Increased sensitivity of asthmatic airway smooth muscle cells to prostaglandin E2 might be mediated by increased numbers of E-prostanoid receptors.  
AU Burgess Janette K; Ge Qi; Boustany Sarah; Black Judith L; Johnson Peter R A  
CS Respiratory Research Group, Department of Pharmacology, Bosch Building D05, University of Sydney, Sydney, NSW 2006, Australia.  
SO The Journal of allergy and clinical immunology, (2004 May) Vol. 113, No. 5, pp. 876-81.  
Journal code: 1275002. ISSN: 0091-6749.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Abridged Index Medicus Journals; Priority Journals  
EM 200406  
ED Entered STN: 20040510  
Last Updated on STN: 20040618  
Entered Medline: 20040617  
AB BACKGROUND: An increase in airway smooth muscle (ASM) cell proliferation leads to an increase in the bulk of the ASM, one of the characteristic features of **asthma**. We have previously shown that ASM cells from asthmatic individuals proliferate more than those from nonasthmatic subjects. This increased growth might be due to compromised inhibitory mechanisms within the ASM of asthmatic subjects. OBJECTIVE: The purpose of this study was to determine whether the proliferative control exerted by prostaglandin E(2) (PGE(2)) was altered in the asthmatic ASM cells. METHODS: We used tritiated thymidine uptake to measure cell proliferation and cell-surface ELISAs to detect the presence of cell-surface receptors on ASM cells isolated from asthmatic and nonasthmatic individuals. RESULTS: The asthmatic ASM cells were significantly more sensitive to proliferation inhibition by PGE(2) than the nonasthmatic cells (P<.02). The PGE(2) (E-prostanoid [EP]) receptors EP2 and EP3 were detected on asthmatic and nonasthmatic smooth muscle cells in culture. There were significantly more receptors on the asthmatic cells. The asthmatic cells also had increased sensitivity to proliferation inhibition by EP2-specific agonists but not by EP3-specific agonists. CONCLUSION: The increased growth observed in asthmatic ASM cells is not the result of impaired responsiveness to PGE(2). In contrast, these cells have increased sensitivity. This increased sensitivity might be mediated by the increased numbers of EP2 receptors on the surface.

L1 ANSWER 5 OF 7 MEDLINE on STN  
AN 2003137787 MEDLINE  
DN PubMed ID: 12618422  
TI Receptors and pathways mediating the effects of prostaglandin E2 on airway tone.  
AU Tilley Stephen L; Hartney John M; Erikson Christopher J; Jania Corey; Nguyen Mytrang; Stock Jeffrey; McNeisch John; Valancius Cathy; Panettieri Reynold A Jr; Penn Raymond B; Koller Beverly H  
CS Department of Medicine, Division of Pulmonary and Critical Care Medicine, University of North Carolina at Chapel Hill, 27599-7248, USA.  
NC HL-04280 (NHLBI)  
HL-58506 (NHLBI)  
HL-68141 (NHLBI)  
SO American journal of physiology. Lung cellular and molecular physiology, (2003 Apr) Vol. 284, No. 4, pp. L599-606. Electronic Publication: 2002-12-13.  
Journal code: 100901229. ISSN: 1040-0605.

CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
LA English  
FS Priority Journals  
EM 200304  
ED Entered STN: 20030326

Last Updated on STN: 20030416

Entered Medline: 20030410

AB Prostaglandin E(2) (PGE(2)) has complex effects on airway tone, and the existence of four PGE(2) [E-prostanoid (EP)] receptors, each with distinct signaling characteristics, has provided a possible explanation for the seemingly contradictory actions of this lipid mediator. To identify the receptors mediating the actions of PGE(2) on bronchomotor tone, we examined its effects on the airways of wild-type and EP receptor-deficient mice. In conscious mice the administration of PGE(2) increased airway responsiveness primarily through the EP1 receptor, although on certain genetic backgrounds a contribution of the EP3 receptor was detected. These effects of PGE(2) were eliminated by pretreatment with either atropine or bupivacaine and were undetectable in anesthetized mice or in denervated tracheal rings, where only EP2-mediated relaxation of airway smooth muscle was observed. Together, our findings are consistent with a model in which PGE(2) modulates airway tone by activating multiple receptors expressed on various cell populations and in which the relative contribution of these receptors might depend on the expression of modifier alleles. PGE(2)/EP1/EP3-induced airway constriction occurs indirectly through activation of neural pathways, whereas PGE(2)-induced bronchodilation results from direct activation of EP2 receptors on airway smooth muscle. This segregation of EP receptor function within the airway suggests that PGE(2) analogs that selectively activate the EP2 receptor without activating the EP1/EP3 receptors might prove useful in the treatment of **asthma**.

L1 ANSWER 6 OF 7 MEDLINE on STN

AN 2002242595 MEDLINE

DN PubMed ID: 11979724

TI The roles of the prostanoids played in the body.

AU Ushikubi Fumitaka; Narumiya Shuh

CS Department of Pharmacology, Asahikawa Medical College, Midorigaoka-Higashi 2-1-1-1, Asahikawa 078-8510, Japan.

SO Nippon yakurigaku zasshi. Folia pharmacologica Japonica, (2002 Apr) Vol. 119, No. 4, pp. 201-7. Ref: 13

Journal code: 0420550. ISSN: 0015-5691.

CY Japan

DT Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

LA Japanese

FS Priority Journals

EM 200206

ED Entered STN: 20020501

Last Updated on STN: 20020619

Entered Medline: 20020618

AB The actions of prostanoids in various physiological and pathophysiological conditions have been examined using mice lacking the prostanoid receptors. PGD2 was found to be a mediator of allergic **asthma**.

Prostaglandin (PG) I2 worked not only as a mediator of inflammation but also as an antithrombotic and cardio-protective agent. Several important actions of PGE2 are brought out via the PGE2-receptor subtype **EP3**

; PGE2 participated in the regulation of platelet function, and it worked as a mediator of febrile responses to both endogenous and exogenous pyrogens. These novel findings on the roles of the prostanoids would contribute to the development of drugs targeting the prostanoid receptors.

L1 ANSWER 7 OF 7 MEDLINE on STN

AN 2000448157 MEDLINE

DN PubMed ID: 11001172

TI Roles of prostanoids revealed from studies using mice lacking specific  
 prostanoid receptors.  
 AU Ushikubi F; Sugimoto Y; Ichikawa A; Narumiya S  
 CS Department of Pharmacology, Asahikawa Medical College, Japan.  
 SO Japanese journal of pharmacology, (2000 Aug) Vol. 83, No. 4, pp. 279-85.  
 Ref: 61  
 Journal code: 2983305R. ISSN: 0021-5198.  
 CY Japan  
 DT Journal; Article; (JOURNAL ARTICLE)  
 General Review; (REVIEW)  
 LA English  
 FS Priority Journals  
 EM 200102  
 ED Entered STN: 20010322  
 Last Updated on STN: 20010322  
 Entered Medline: 20010202  
 AB The actions of prostanoids in various physiological and pathophysiological  
 conditions have been being examined using mice lacking different  
 prostanoid receptors. Prostaglandin (PG) I2 worked not only as a mediator  
 of inflammation but also as an antithrombotic agent. PGF2alpha was found  
 to be an essential inducer of labor. Several important actions of PGE2  
 are exerted via each of the four PGE2 receptor subtypes: EP1, EP2,  
 EP3 and EP4. PGE2 participated in colon carcinogenesis via the  
 EP1. PGE2 also participates in ovulation and fertilization and  
 contributes to the control of blood pressure under high-salt intake via  
 the EP2. PGE2 worked as a mediator of febrile responses to both  
 endogenous and exogenous pyrogens and as a regulator of bicarbonate  
 secretion induced by acid-stimulation in the duodenum via the EP3  
 . It regulated the closure of ductus arteriosus and showed bone resorbing  
 action via the EP4. PGD2 was found to be a mediator of allergic  
 asthma. These studies have revealed important roles of  
 prostanoids, some of which had not previously been known.

=>

---Logging off of STN---

=>

Executing the logoff script...

=> LOG Y

COST IN U.S. DOLLARS	SINCE FILE	TOTAL
	ENTRY	SESSION
FULL ESTIMATED COST	7.00	7.42

STN INTERNATIONAL LOGOFF AT 12:06:12 ON 05 APR 2006